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Echocardiography and rupture of the heart

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SUMMARY In four patients with rupture of the free wall of the left ventricle, the diagnosis was made at the bedside using a portable cross-sectional echocardiograph; this was confirmed at necropsy in two patients and by paracentesis in the other two.

Weak ventricular contractions were seen after each QRS complex of the electrocardiogram, contradicting a widespread belief that cardiac rupture immediately produces electromechanical dissociation. In one patient aspiration of 100 ml blood from the pericardium allowed resumption of cardiac pump function; this patient survived for three days but was allowed to die because of cerebral damage sustained during resuscitation.

Rupture of the free wall of the left ventricle is usually unheralded, occurring in one to five per cent of all myocardial infarctions; at present this represents 20 per cent of in-hospital mortality for these patients.[1–3]

The clinical diagnosis of acute cardiac rupture is suggested by the absence of pulse and heart sounds despite a normal electrocardiographic monitoring lead.[4]

During the past few months we used echocardiography at the bedside in four patients with cardiac rupture. The instrument (Minivisor, Organon Technika) is a mushroom-shaped, portable, battery-powered, linear array echocardiograph which displays a 10×20 cm cross-section of the heart on a 2×4 cm oscilloscope[5] but does not make graphic recordings.

Case reports

CASE 1

A 66-year-old man suddenly became comatose three days after an acute transmural anterior myocardial infarction. The pulse could not be felt even in the carotid arteries, yet electrocardiographic monitoring showed normal sinus rhythm. Immediate echocardiographic examination showed that the long axis cross-section of the heart was surrounded by a characteristic horseshoe-shaped contour of echo-free pericardial effusion[6]; the left ventricle was very dilated, and each QRS complex of the electrocardiogram was associated with hypokinetic cardiac contractions. Blood was present in the pericardium. This patient was prounced dead after five minutes of attempted resuscitation (wide fixed pupils and electrocardiographic as well as echocardiographic cardiac standstill). Necropsy was refused by the family.

CASE 2

A 58-year-old man experienced chest discomfort on the fifth day after an uncomplicated transmural diaphragmatic myocardial infarction; 10 minutes later he suddenly lost consciousness. Weak carotid and femoral pulses could be felt for 45 minutes. Echocardiography showed a dilated left ventricle and left atrium with hypokinetic movements of the posterior wall of the left ventricle and no movements of the interventricular septum. The cardiac silhouette was surrounded by a horseshoe-shaped crescent of echo-free pericardial fluid; pericardiocentesis disclosed unclotted blood. Despite continued attempts at resuscitation, the carotid pulses disappeared five minutes before the onset of an agonal rhythm on the electrocardiographic monitor. At necropsy, a cardiac tear was found at the border of the transmural infarct, with 250 ml blood in the pericardial sack.

CASE 3

A 75-year-old man sustained pulmonary oedema and ventricular fibrillation shortly after the onset of acute diaphragmatic infarction; resuscitation was

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successful. On the second day he complained of chest pain which responded well to glyceryl trinitrate. On the third day he experienced epigastric pain, and 30 minutes later developed pulmonary oedema and ventricular fibrillation. Defibrillation restored sinus rhythm but not blood pressure. Echocardiography showed pericardial effusion and movements of the mitral valve associated with each QRS complex. Pericardial puncture showed unclotted blood at a depth of 3 cm from the skin surface; 100 ml blood was aspirated from the pericardium, and, progressively, the blood pressure reappeared, diuresis resumed, and peripheral circulation reverted to normal. At that time, the echo-free horseshoe-shaped pericardial effusion had disappeared, the left ventricle was enlarged with good contractions of the interventricular septum; the posterior wall of the left ventricle was poorly visualised and appeared akinetic. Unfortunately, brain damage occurred during the lengthy resuscitation procedure (one hour before normalisation of blood pressure) and the patient died four days later. Necropsy was refused by the family.

CASE 4

A 73-year-old man was recovering well from a lateral myocardial infarction when, on the sixth day after his infarct, he suddenly collapsed on the ward. Resuscitation was initiated within 30 seconds; the electrocardiogram showed slow sinus rhythm but the carotid pulse was impalpable. Echocardiography showed an enlarged left ventricle with weak contractions of the ventricular walls and movements of the anterior mitral valve leaflet; there was a horseshoe-shaped crescent of pericardial effusion. Resuscitation failed and the patient was pronounced dead 30 minutes later. Necropsy showed obstruction of the left circumflex artery with a recent lateral myocardial infarction, a tear through the infarcted area, and 300 ml blood in the pericardium.

Discussion

Only immediate cardiac surgery with repair of the tear in the free wall of the left ventricle is likely to save the life of patients with rupture of the heart.[2] Few interventions have been successful in cardiac rupture,[7–9] but O'Rourke[10] reported a more favourable outcome when an early diagnosis was made. However, cardiac surgery is hazardous soon after an acute myocardial infarction, so this risk is acceptable only if the diagnosis of cardiac rupture is certain. This certainty can be provided within 30 seconds by means of a portable cross-sectional echocardiograph.

The absence of pulse and heart sounds despite normal sinus rhythm on the electrocardiogram has suggested that cardiac rupture after acute myocardial infarction produces tamponade and electromechanical dissociation. Cross-sectional echocardiography showed that each QRS complex was associated with weak contractions of the non-infarcted walls of the heart, even after the onset of an agonal rhythm. Electromechanical coupling is still present in these patients, even though the resulting movements of the left ventricular walls produce no palpable peripheral pulses.

Aspiration of 100 ml pericardial blood was associated with a resumption of cardiac pump function in one patient. One wonders whether invasive resuscitation might save some patients with a ruptured left ventricle.

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